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CARDIAC FUNCTION AND HEART FAILURE

HIGH SENSITIVITY C-REACTIVE PROTEIN AS AN INDEPENDENT PREDICTOR OF PROGRESSIVE MYOCARDIAL FUNCTIONAL DETERIORATION: THE LONGITUDINAL MULTI-ETHNIC STUDY OF ATHEROSCLEROSIS

ACC Poster Contributions
Ernest N. Morial Convention Center, Hall F
Sunday, April 03, 2011, 3:30 p.m.-4:45 p.m.

Session Title: Biomarkers and Prognostication in Heart Failure
Abstract Category: 24. Myocardial Function/Heart Failure–Clinical Nonpharmacological Treatment
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Objectives: In this study, we used longitudinal MRI follow-up analysis to examine whether C-reactive protein (CRP) levels relate to progressive myocardial functional deterioration as a potential mechanism of incident heart failure.

Background: Systemic inflammation has been linked to the development of heart failure in population studies including MESA (Multi-Ethnic Study of Atherosclerosis) but little evidence exists regarding potential mechanism of this relationship. Method: Regional myocardial functional data from MESA participants who had baseline CRP measurement and also underwent tagged cardiac MRI both at baseline and at five-year follow-up (n=785) were analyzed. Left ventricular (LV) midwall peak systolic circumferential strain (Ecc), of which a more negative value denotes stronger regional myocardial function, was measured, while Ecc change was calculated as the difference between baseline and follow-up Ecc.

Results: During the follow-up period, participants with elevated CRP experienced a decrease in strain, independent of age, gender and ethnicity (B=0.08; ΔEcc change per 1mg/L CRP change, 95% CI 0.04-0.13, p<0.001, Model 1), and additionally beyond systolic blood pressure, diabetes, smoking status, body mass index, current medication and glomerular filtration rate (B=0.10, 95% CI 0.05-0.14, p<0.001, Model 2). The relationship remained statistically significant after further adjustment for LV mass, coronary calcium score and interim clinical coronary events (B=0.10, 0.05-0.15, p<0.001).

Conclusions: Higher CRP levels are related to progressive myocardial functional deterioration independent of subclinical atherosclerosis and clinical coronary events in asymptomatic individuals without previous history of heart disease.